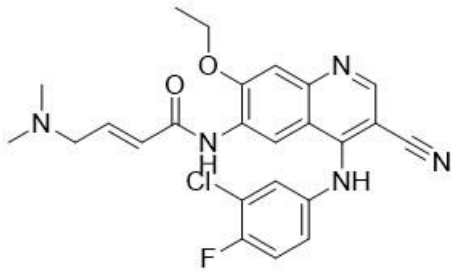
Product data sheet

MedKoo Cat#: 202190 Name: Pelitinib CAS#: 257933-82-7 Chemical Formula: $C_{24}H_{23}ClFN_5O_2$ Exact Mass: 467.15243 Molecular Weight: 467.92	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Pelitinib, also known as EKB569 and WAY-172569, is a 3-cyanoquinoline pan-ErbB tyrosine kinase inhibitor with potential antineoplastic activity. EKB-569 irreversibly binds covalently to epidermal growth factor receptors (EGFR) ErbB-1, -2 and -4, thereby inhibiting receptor phosphorylation and signal transduction and resulting in apoptosis and suppression of proliferation in tumor cells that overexpress these receptors.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	10.0	21.4

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.14 mL	10.69 mL	21.37 mL
5 mM	0.43 mL	2.14 mL	4.27 mL
10 mM	0.21 mL	1.07 mL	2.14 mL
50 mM	0.04 mL	0.21 mL	0.43 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Kim H, Lim HY. Novel EGFR-TK inhibitor EKB-569 inhibits hepatocellular carcinoma cell proliferation by AKT and MAPK pathways. J Korean Med Sci. 2011 Dec;26(12):1563-8. doi: 10.3346/jkms.2011.26.12.1563. Epub 2011 Nov 29. PMID: 22147992; PMCID: PMC3230015.

2. Aravindan N, Thomas CR Jr, Aravindan S, Mohan AS, Veeraraghavan J, Natarajan M. Irreversible EGFR inhibitor EKB-569 targets low-LET γ-radiation-triggered rel orchestration and potentiates cell death in squamous cell carcinoma. PLoS One. 2011;6(12):e29705. doi: 10.1371/journal.pone.0029705. Epub 2011 Dec 29. PMID: 22242139; PMCID: PMC3248439.

In vivo study

1. Crabtree JE, Jeremy AH, Duval C, Dixon MF, Danjo K, Carr IM, Pritchard DM, Robinson PA. Effects of EGFR Inhibitor on Helicobacter pylori Induced Gastric Epithelial Pathology in Vivo. Pathogens. 2013 Oct 14;2(4):571-90. doi: 10.3390/pathogens2040571. PMID: 25437333; PMCID: PMC4235704.

7. Bioactivity

Biological target:

Pelitinib (EKB-569;WAY-EKB 569) is an irreversible inhibitor of EGFR with an IC50 of 38.5 nM

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In vitro activity

The purpose of this in vitro study was to investigate the effects of the second-generation compound (EKB-569) in HCC. EKB-569 was evaluated for its potential as part of a chemosensitizing combination treatment with sorafenib, in tailored therapies for resistant tumors. Thirteen exponentially growing HCC cell lines were trypsinized and plated at 4-5 × 10^3 cells/mL in 96-well culture plates. After 24 hr incubation, cells were treated for 72 hr with serial three-fold dilutions of 10 μM for each drug. In the results from 13 HCC cell lines, the novel irreversible EGFR inhibitor EKB-569 showed higher efficacy than first generation, reversible EGFR inhibitors (Table 1). In SK-Hep1 cells, after treatment with EKB-569, we found down-regulation of phosphorylated ERK and AKT and up-regulation of p21 in a dose-dependent manner. Furthermore, the combination of sorafenib and EKB-569 might be able to overcome HCC resistance to EGFR inhibitors. It has been shown for the first time that sorafenib plus EKB-569 has an effect on drug-resistant cell lines.

Reference: J Korean Med Sci. 2011 Dec; 26(12): 1563–1568.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3230015/

In vivo activity

To examine the importance of EGFR signalling to gastric pathology, this study investigated whether treatment of Mongolian gerbils with a selective EGFR tyrosine kinase inhibitor, EKB-569, altered gastric pathology in chronic H. pylori infection. Gerbils were infected with H. pylori and six weeks later received either EKB-569-supplemented, or control diet, for 32 weeks prior to sacrifice. EKB-569-treated H. pylori-infected gerbils had no difference in H. pylori colonisation or inflammation scores compared to infected animals on control diet, but showed significantly less corpus atrophy, mucous metaplasia and submucosal glandular herniations along with markedly reduced antral and corpus epithelial proliferation to apoptosis ratios. EKB-569-treated infected gerbils had significantly decreased abundance of Cox-2, Adam17 and Egfr gastric transcripts relative to infected animals on control diet. EGFR inhibition by EKB-569 therefore reduced the severity of pre-neoplastic gastric pathology in chronically H. pylori-infected gerbils. EKB-569 increased gastric epithelial apoptosis in H. pylori-infected gerbils which counteracted some of the consequences of increased gastric epithelial cell proliferation. Similar chemopreventative strategies may be useful in humans who are at high risk of developing H. pylori- induced gastric adenocarcinoma.

Reference: Pathogens. 2013 Dec; 2(4): 571–590.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4235704/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.